Threshold Vision in Amblyopia: Orientation and Phase

Robert F. Hess[1,2] *and Sam A. Malin*[3]

Purpose. The visual deficit in amblyopia involves both elevated contrast thresholds and distorted suprathreshold percepts at high spatial frequencies. It is currently unclear whether these two anomalies are part of the same neural disturbance or whether they reflect different neural dysfunction.

Methods. The quality of the spatial percepts in amblyopia was assessed at detection threshold. The ability of amblyopes to discriminate the orientation and local spatial phase of well-localized spatial stimuli was measured at the detection threshold. Measurements were made as a function of spatial frequency.

Results. Performance seemed normal for orientation discrimination, even at high spatial frequencies, but, in some cases, it was disturbed in phase discrimination.

Conclusions. A different explanation and neural basis is needed to encompass both threshold and suprathreshold spatial deficits in amblyopia. (*Invest Ophthalmol Vis Sci.* 2003;44: 4762–4771) DOI:10.1167/iovs.03-0259

Amblyopia is a uniocular impairment of vision resulting from strabismus, anisometropia, or form deprivation occurring early in visual development. Initially, the deficit in amblyopia was quantified solely in terms of how contrast thresholds were raised across a number of stimulus dimensions—chiefly, spatial frequency,[1–3] eccentricity,[4] and luminance.[5] Several subsequent findings led to a broadening of our view of the amblyopic deficit. First, contrast perception above threshold was normal.[6] Second, amblyopes experience spatial distortions with suprathreshold stimuli.[7–12]

Thus, there appear to be two distinct perceptual anomalies: elevated contrast thresholds at high spatial frequencies and distorted percepts of high spatial frequency stimuli at suprathreshold contrasts. The relationship between these two perceptual anomalies, however, is not currently known in humans. Animal models have not been able to shed any light on this issue. Neurophysiological studies in animals made artificially amblyopic (anisometropic or strabismic) have suggested that cortical cells in area V1 responding to high spatial frequencies have elevated thresholds when driven by the amblyopic eye, thus providing an explanation for the contrast threshold deficit in humans.[13–16] However, no anomalies have been found in the spatial (e.g, orientation tuning or receptive field geometry) properties (other than their reduced resolution) of individual cortical cells driven by the amblyopic eye that might provide any insight into an explanation for the reported suprathreshold distortions.[13–16]

In an effort to understand the relationship between the elevated thresholds and the suprathreshold spatial distortions in amblyopia, we asked whether these distorted percepts were present at contrasts at which the detection thresholds are elevated. To investigate, we measured two different types of spatial discriminations at contrasts corresponding to detection thresholds across a wide range of spatial frequencies. Because the previously reported suprathreshold spatial distortions reported by amblyopes have been considered as either an orientation-specific[17,12] or spatial phase-specific[18–20] problem, we used both orientation and phase measures for our discrimination task. We used stimuli that are well-localized in space and spatial frequency, consistent with the known properties of cortical receptive fields. The results suggest that amblyopes can accurately discriminate orientation differences at detection threshold, suggesting that local orientation information carried by cells with raised contrast thresholds is undisturbed. Some amblyopes exhibit problems with local phase discrimination at detection threshold. Thus, cells with elevated thresholds can exhibit normal orientation but abnormal phase processing. The orientation anomaly in amblyopia evident at suprathreshold levels may have a separate explanation from that of the contrast sensitivity deficit and the related phase-processing deficit.

Methods

Apparatus

All stimuli were generated and presented and the response collected and analyzed on a minicomputer (11/34A PDP; Digital Equipment Corp. [no longer produced]). The stimuli were presented in a Joyce Electronics video monitor (raster display; 30 cm × 20 cm with a white P4 phosphor; Cambridge, Cambridge, UK) through an interface (Design CED 502; Cambridge Electronics). The mean screen luminance was 100 cd/m^2 and the frame rate was 100 Hz in most experiments. The contrast linearity of the display screen was measured and found to hold up to 98% contrast.

Stimulus

Horizontally oriented sinusoidal grating patterns, in which contrast was modulated sinusoidally in time were used to measure thresholds for contrast detection. The choice of the horizontal orientation ensured that any unsteadiness of the eye (predominantly in the horizontal plane) would not interfere with measurement accuracy by introducing retinal image smear.[21]

The patterns were generated digitally. According to the method of Robson and Graham[22] the contrast of each stimulus was weighted with Gaussian functions of space and time (x, y, t). This ensured that the stimuli were well localized spatially and temporally.

The luminance distribution of each stimulus was specified by

$$L(x,y,t) = L_o [1 + CG(x,y,t) \times \sin (2\pi f_x X] \quad (1)$$

where L_o indicates the space-averaged luminance, C the contrast variable, and f_x the spatial frequency. The window (spread) function was given by

$$G(x,y,t) = \exp[-(x/S_x)^2 - (y/S_y)^2 - (t/S_t)^2] \quad (2)$$

From the [1]McGill Vision Research, Department of Ophthalmology, McGill University, Montreal, Quebec, Canada; [2]The School of Optometry, Queensland University of Technology, Brisbane, Queensland, Australia; and [3]The Physiological Laboratory, Cambridge, United Kingdom.

Supported by Canadian Institutes of Health Research Grant MT108-18 (RFH).

Submitted for publication March 13, 2003; accepted April 22, 2003.

Disclosure: **R.F. Hess**, None; **S.A. Malin**, None

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be marked "*advertisement*" in accordance with 18 U.S.C. §1734 solely to indicate this fact.

Corresponding author: Robert F. Hess, McGill Vision Research, Department of Ophthalmology, McGill University, 687 Pine Ave W. (H4-14), Montreal, Quebec, Canada H3A 1A1; robert.hess@mcgill.ca.

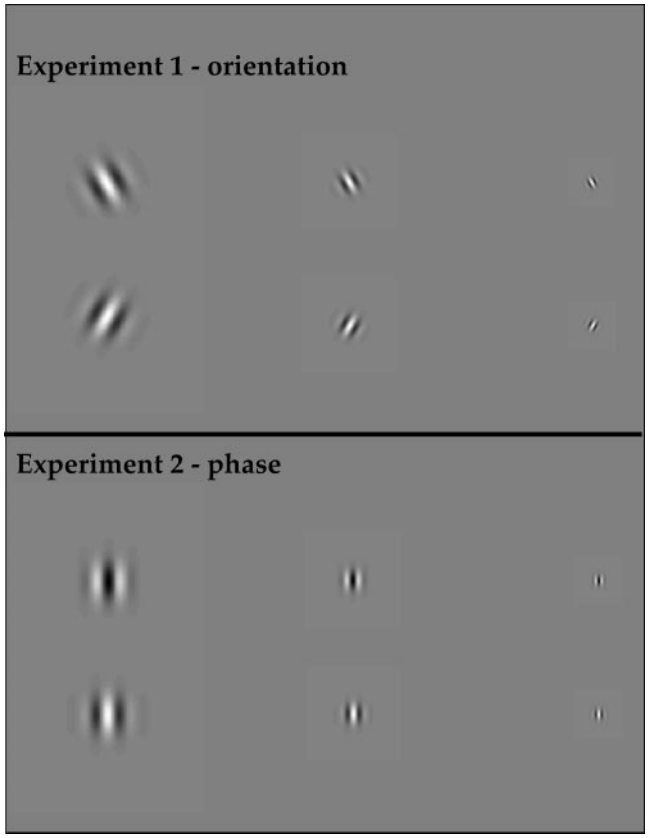

FIGURE 1. Gabor stimuli used for orientation (*top*) and phase (*bottom*) detection/discrimination. Three different spatial frequencies are displayed.

The term "spread" signifies the distance in time or space wherein the Gaussian falls from 1 to 1/*e* (approximately 0.37). The overall spread function is the product of the horizontal, vertical, and time Gaussians, with the spreads S_x, S_y, and S_t, respectively. The spatial and temporal windows were each truncated at plus and minus two spreads.

The Gaussian weighting of the stimuli localized the patterns in spatial and temporal frequency terms; the Gaussian amplitude spectra were centered on f_x and f_t, with spreads of $1/(\pi S_x)$ and $1/(\pi S_t)$, respectively. The overall spatial Gaussian spread was circular ($S_x = S_y$) and set to 0.5 periods of the test spatial frequency; consequently, the spatial frequency bandwidth (full width at half-amplitude) for each stimulus pattern was fixed at 1.1 octaves. The temporal Gaussian spread was set to 250 ms.

Seven spatial frequencies over a range of 8 octaves above 0.3 cyc/deg were tested. The one-dimensional stimuli were generated in cosine phase relative to the screen center (i.e, there was a bright or dark bar at the pivotal center of each pattern). The appearance of these stimuli on the display screen at highly suprathreshold contrast is shown in Figure 1.

General Procedure

The video display was mounted at eye level in a fixed location immediately in front of the subject and was laterally centered on his test eye. The display screen was surrounded by a large luminance-matched field that subtended 80° horizontally by 60° vertically at the usual viewing distance of 3.7 m.

Psychophysical Paradigm

A temporal, two-alternative, forced-choice (2-AFC) technique, with feedback, was used to minimize subjective test bias: the interactive staircase procedure was driven by the subject's responses and con-

TABLE 1. Amblyope Group

Subject	Sex	Class	Clinical Data (Acuity)	Clinical History
LC	M	S	15° L. EXT Central fix. Rx R−1.50/−0.75×180 (20/10) L−1.50/−0.50×180 (20/80)	First Rx age 24 y; no surgery/orthop. trt.
AF	F	S	R. ESOT (part accm) 0.75° ecc. fix. Rx R−1.25/−1.0×10 (20/60) L−2.50/−1.50×170 (20/10)	First Rx age 3 y; occln. therapy
CF	M	S	L. EXOT 15° ecc. fix. Rx R+4.50/−0.50×180 (20/10) L+4.25/DS (20/600)	First Rx age 6 y; surgery 8 y
CG	M	S-A	L. ESOμT 0.5° ecc. fix. Rx R plano (20/10) L+3.50/−1.00×90 (20/400)	First Rx age 6 y; occln. therapy
JS	F	S-A	R. EXOμT Central fix. Rx R−5.00DS (20/400) L+1.00/−0.75×15 (20/10)	No Rx as child; no surgery/therapy
ST	F	S	L. ESOT 10° ecc. fix. Rx R−0.25/−0.50×110 (20/10) L+0.50DS (20/1800)	First Rx age 5 y; occln. therapy
NN	M	A	Central fix. Rx Rplano/−.50×105 (20/10) L−1.75/−3.00×10 (20/80)	First Rx age 25 y; no orthop. therapy
SM	M	A	Central fix. Rx R−0.50 (20/20) L+3.00 (20/60)	No Rx worn; no orthop. therapy

S, strabismic; A, anisometropic; S-A, strabismic and anisometropic; L, left eye; R, right eye; EXT, exotropia; ESOT, esotropia; μ, microtropia; ecc fix, eccentric fixation; Rx, optical correction; orthop trt, orthopic treatment; occln, occlusion; part accm, partly accommodative squint.

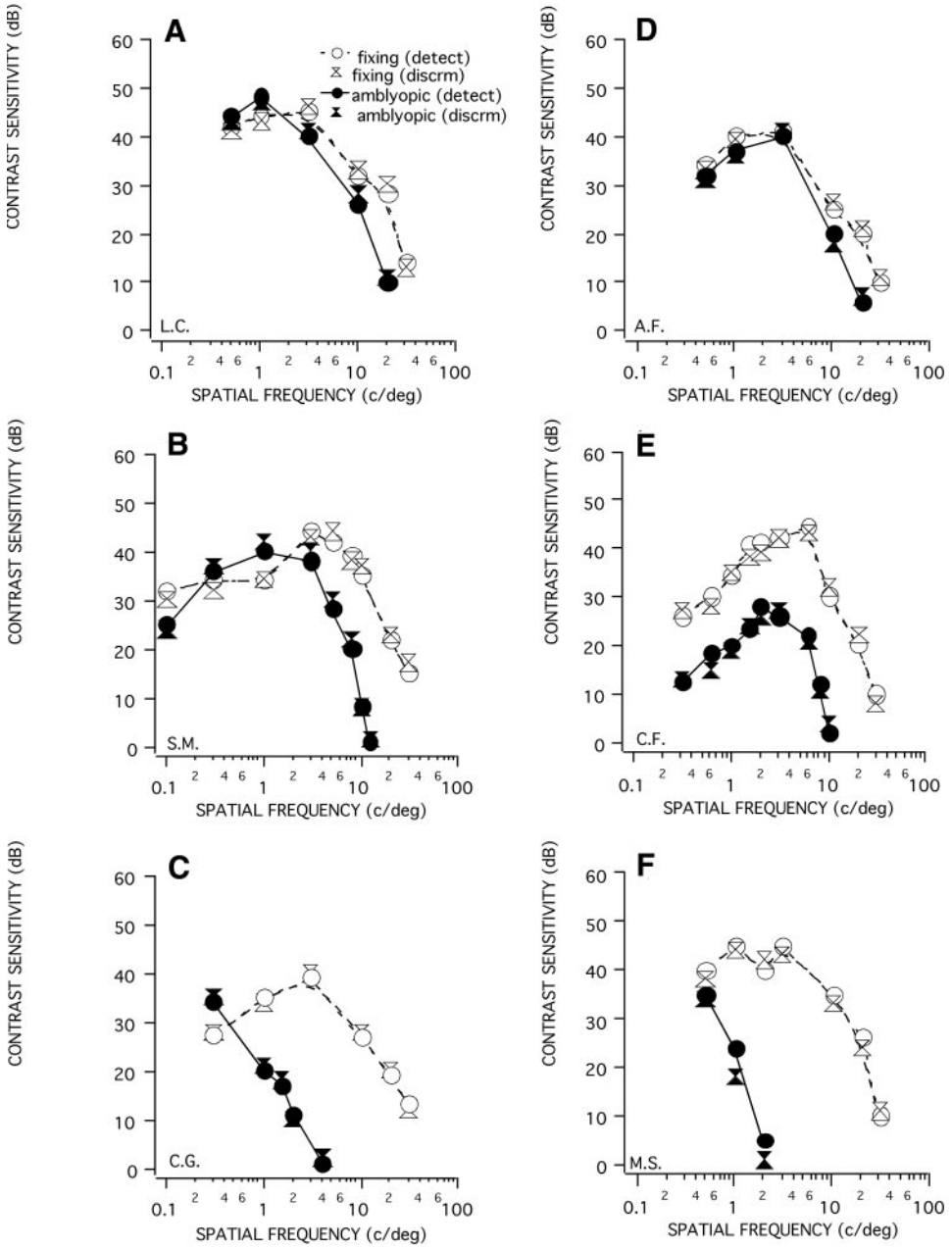

FIGURE 2. Comparison of the contrast needed to detect (*circular symbols*) and to discriminate (*hourglass symbols*) the orientation (70° vs. 110°) of a Gabor stimulus as a function of spatial frequency for the fixing and fellow amblyopic eyes of six amblyope observers. Standard deviations are smaller than the symbol sizes.

trolled by computer. Each trial consisted of two presentations (denoted by auditory tones). In experiment 1, for the detection task, the stimulus was a Gabor oriented at 70° or 110° (chosen at random) paired with a screen of the same average luminance. The subject had to choose which interval contained the Gabor (the contrast detection threshold). For the discrimination task, a similar procedure was used, but this time the Gabor oriented at 70° was paired with the Gabor oriented at 110°. The subject had to choose which interval contained the Gabor oriented at 70° (the orientation threshold). In experiment 2, a similar technique was used to measure the detection and discrimination of Gabor stimuli of opposite contrast polarity (180° phase difference). Presentation time was limited (S_t = 250 ms), in an attempt to minimize the influence on thresholds of any saccadic eye movements. Immediately after a response had been made, the next two-interval trial started, stimulus contrast being the variable between trials. The average of six reversals of the staircase constituted one mean. Each data point displayed in the figures comprises the arithmetical average of at least three means. The standard deviation was equal to the symbol size and never greater than twice the symbol size.

Clinical Subjects

Eight amblyopes were tested (see Table 1). Each subject underwent a complete orthoptic and ophthalmic examination. All were experienced psychophysical observers, having participated in a number of previous studies of visual function. Informed consent was obtained from all participating subjects after the nature and purpose of the study had been explained. The research adhered to the tenets of the Declaration of Helsinki.

RESULTS

Figure 2 shows a comparison, for six amblyopes, of the contrast necessary to detect (circular symbols) the Gabor stimulus, compared with that necessary to distinguish its orientation (hourglass symbols). It is clear in all cases that the orientation discrimination (70° vs. 110°) and the contrast-detection thresholds were comparable in the fellow fixing (unfilled symbols) and amblyopic eyes (filled symbols). This was true even in

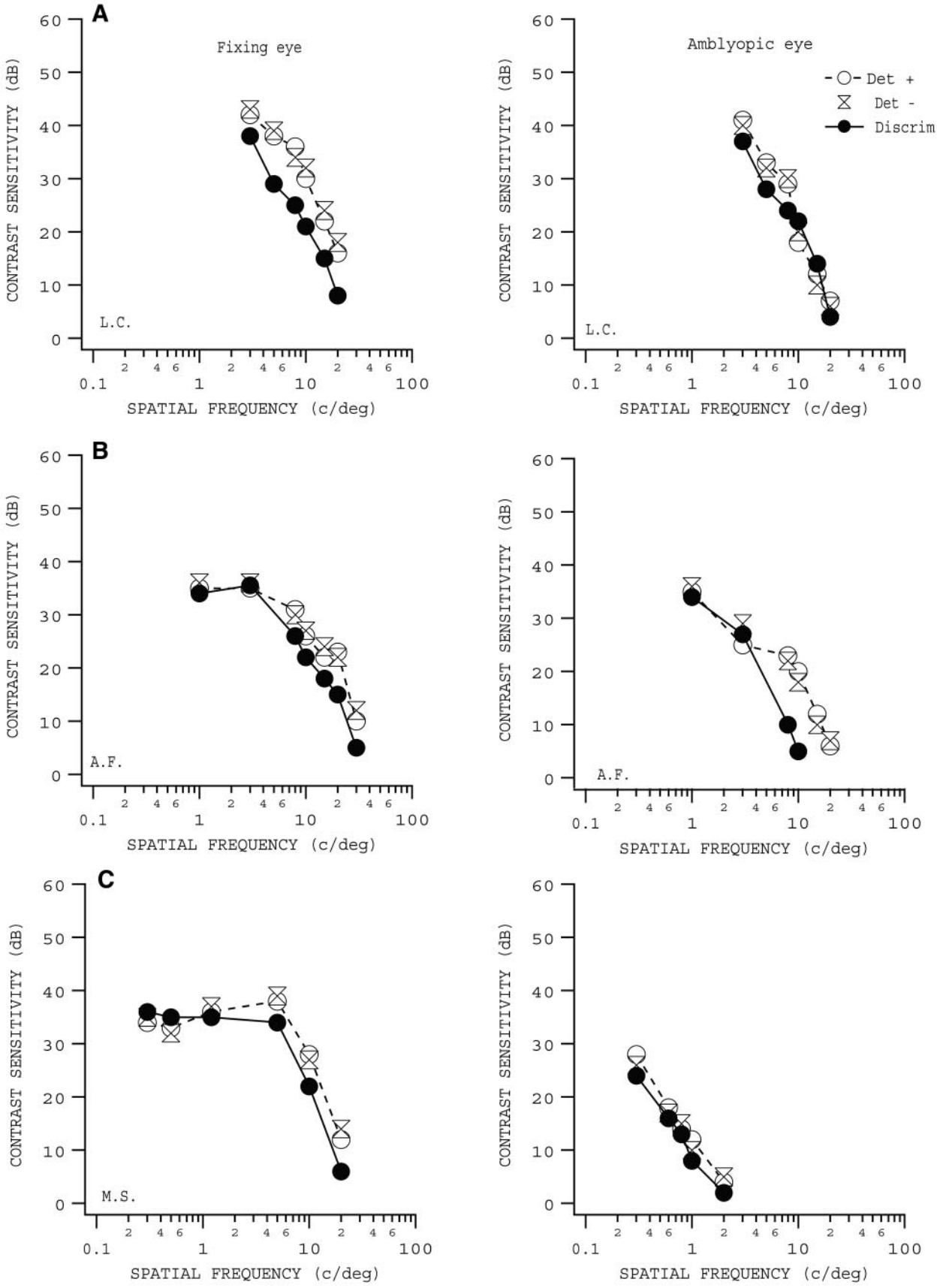

FIGURE 3. Comparison of the contrast needed to detect and to discriminate the phase (0° vs. 180°) of a Gabor stimulus as a function of spatial frequency for the fixing (*left column*) and fellow amblyopic eyes (*right column*) of three amblyopes. Standard deviations are smaller than or equal to the symbol sizes.

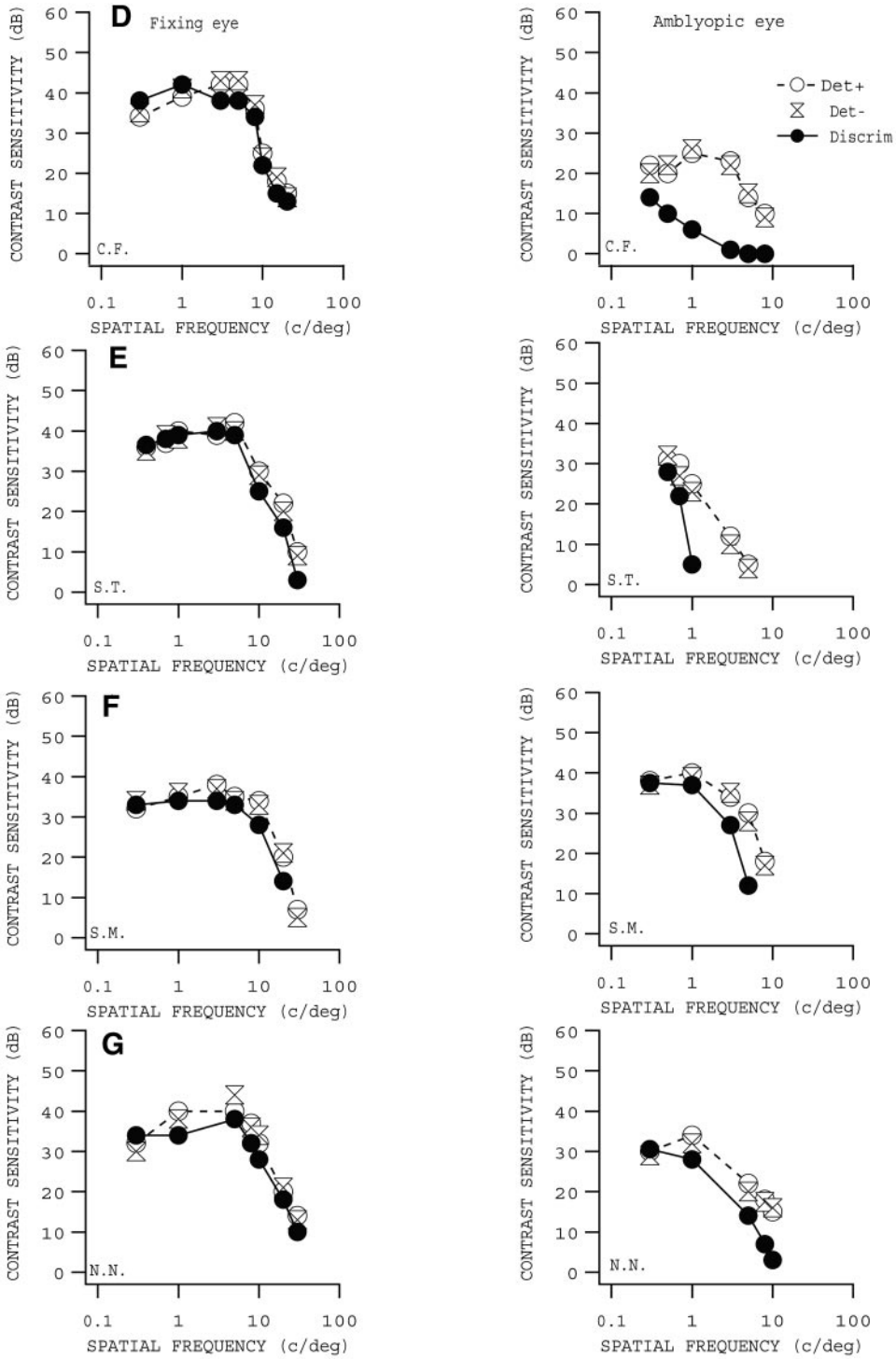

FIGURE 4. Comparison of the contrast needed to detect and to discriminate the phase (0° vs. 180°) of a Gabor stimulus as a function of spatial frequency for the fixing (*left column*) and fellow amblyopic eyes (*right column*) of four amblyopes. Standard deviations are smaller than or equal to the symbol sizes.

amblyopic eyes at spatial frequencies at which contrast thresholds were severely elevated. Similar results were found in normal observers (data not displayed).

The result of a similar comparison for discriminating the local polarity (i.e, 180° phase difference) of the stimulus at detection threshold is shown in Figures 3 and 4. In each of these figures, detection thresholds for positive and negative Gabor stimuli (unfilled symbols) are compared with the contrast need to discriminate these stimuli (filled symbols). Results are shown for the fellow fixing eye (left column) and the amblyopic eye (right column). For the fellow fixing eye, this discrimination can be made reliably at the detection threshold at low but not at high spatial frequencies. At high spatial frequencies slightly more contrast is necessary to discriminate the polarity of the stimuli compared with that necessary to detect them. This was also the case in normal observers (data not shown) and is in accordance with previous studies in subjects with normal vision, using more spatially broadband stimuli.[23] The results displayed in Figure 3 are for a group of subjects with amblyopia of various severities. Two amblyopes, including one with the most severe loss (Fig. 3C), exhibited normal phase discrimination. Another amblyope (Fig. 3B) exhibited anomalous phase discrimination at high spatial frequencies. The results in Figure 4 are for amblyopes for whom more contrast was needed to perform the phase discrimination with the amblyopic eye compared with the fellow fixing eye. This

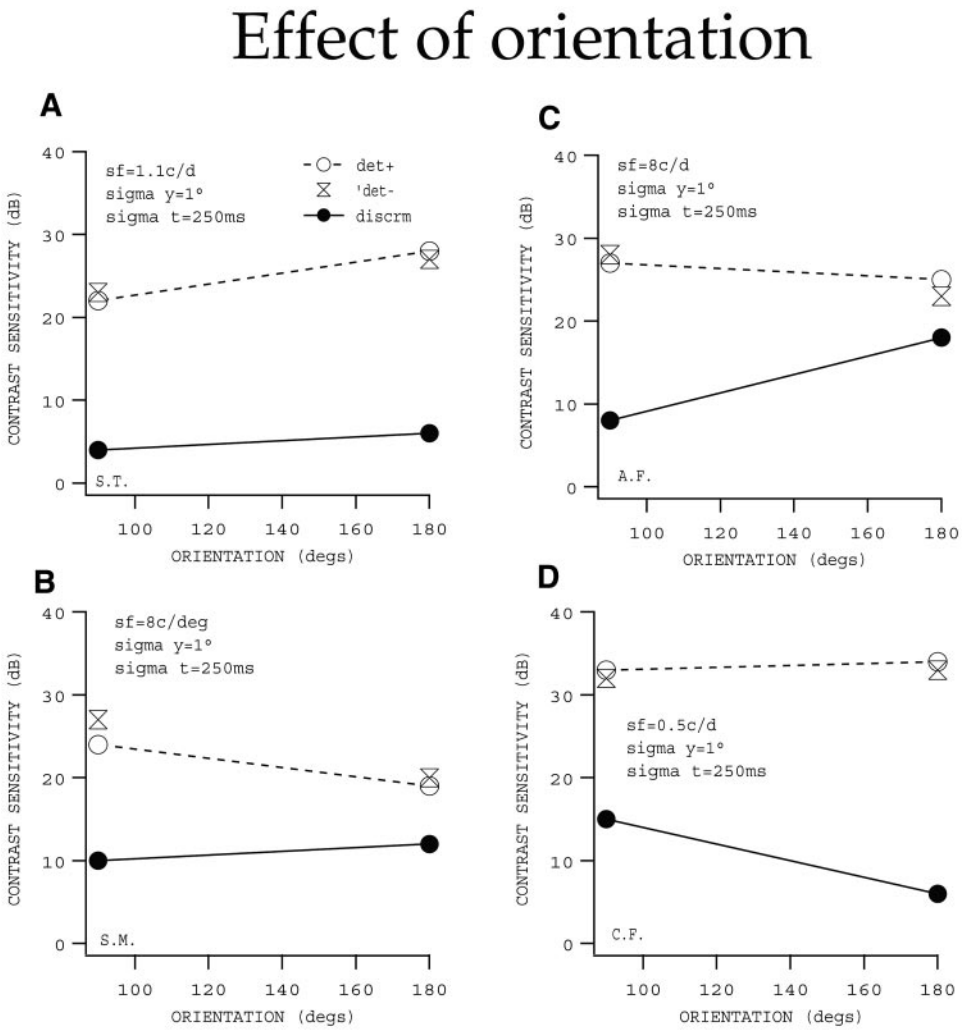

FIGURE 5. The effect of stimulus orientation on the phase discrimination anomaly for a selected high spatial frequency (relative to the amblyopic cutoff acuity) for four amblyopic eyes. Detection is compared with discrimination. Standard deviations are smaller than or equal to the symbol sizes.

discrimination deficit in these cases is worse at high spatial frequencies. For some subjects there was a range of high spatial frequency stimuli for which this discrimination could not be made at any contrast.

In four of the amblyopes who exhibited phase discrimination anomalies, we investigated the effects of absolute orientation, bar length, and duration of presentation. In all cases, we chose stimuli with spatial frequencies that were in the region in which anomalous discrimination had already been demonstrated (Figs. 3, 4). These results are shown in Figures 5 6, and 7, in which the detection thresholds for positive and negative Gabors are compared with discrimination thresholds. Results are shown only for the amblyopic eye.

In terms of the orientation of the stimulus, the original results were obtained with a horizontally oriented Gabor to minimize any effects of eye movements.[21] How important is the absolute orientation? The fellow fixing eye displayed the expected relation with orientation—namely, both detection and discrimination thresholds were, to a first approximation, independent of the stimulus orientation (data not shown). The results in Figure 5 show individual differences for stimulus orientation for amblyopic eyes. The results for one amblyope did not depend critically on the stimulus orientation (subject ST) whereas those for three others (AF, SM, and CF) did. Amblyopes AF and SM who displayed only small deficits in phase discrimination in the original experiments for horizontally oriented stimuli (Figs. 3, 4) exhibited larger deficits for vertically oriented stimuli (Fig. 5). Amblyope CF exhibited a greater discrimination deficit for a horizontal stimulus (Fig. 5).

In terms of bar length, the stimulus used in the original measurements (Figs. 3, 4) had a vertical sigma of 0.5 times the spatial wavelength. To see what effect this has, we measured detection and discrimination performance of the amblyopic eye for shorter ($0.1 \times \lambda$) and longer bars ($1 \times \lambda$). These results are shown in Figure 6. The normal fellow eye exhibited the expected result: Both detection and discrimination thresholds increased to approximately the same extent as the bar length was increased (data not displayed). In the amblyopic eyes, detection sensitivity improved with increasing bar length but discrimination threshold either remained constant (CF) or deteriorated (ST, AF, and SM).

In terms of presentation time, the original measurements (Figs. 3, 4) were obtained with a temporal sigma of 250 ms. Figure 7 shows results for the four amblyopes for longer (500 ms) and shorter (63 ms) durations. The fellow fixing eye exhibited the expected result, namely that both detection and discrimination sensitivity increased to approximately the same extent with increasing presentation duration. The amblyopic results either showed the expected gain in sensitivity for detection and discrimination (CF, SM), or discrimination sensitivity remained constant (ST, AF) for the duration.

DISCUSSION

The present results suggest that amblyopes can correctly discriminate the orientation of a local, spatially band-pass stimulus at detection threshold over the entire spatial range used by the

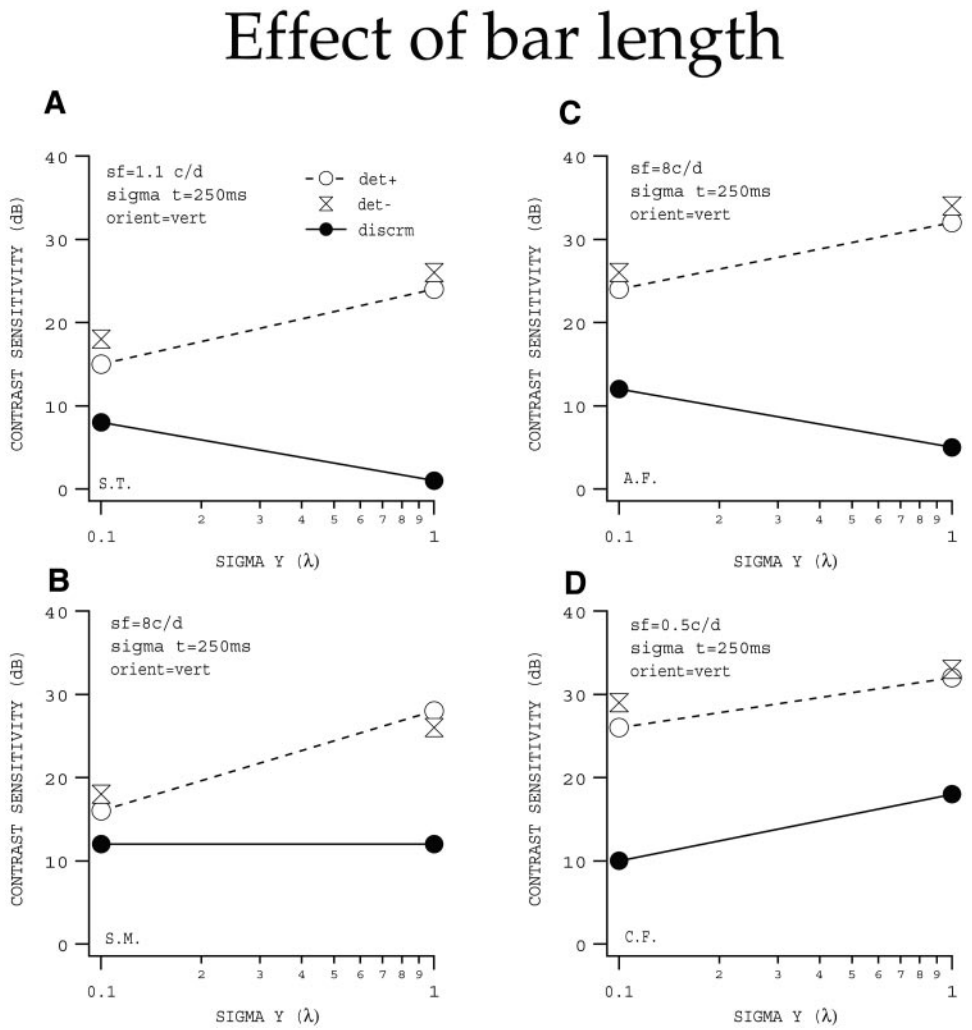

FIGURE 6. The effect of stimulus bar length (sigma *y* of Gaussian space constant in units of the spatial period of the Gabor) on the phase discrimination anomaly for a selected high spatial frequency (relative to the amblyopic cutoff acuity) for four amblyopic eyes. Detection is compared with discrimination. Standard deviations are smaller than the symbol sizes.

amblyopic eye. This is consistent with the results of our earlier study that found that amblyopes could correctly identify the orientation of a high-spatial-frequency grating at the acuity limit.[24] This in turn argues for normal orientation processing at low contrasts at which contrast sensitivity is impaired. Several studies have identified a problem in orientation discrimination in amblyopia at suprathreshold contrasts[17,25,26] that is limited to high spatial frequencies.[27] Such a deficit could in principle be caused by either orientation detectors of broader bandwidth or to a disruption in how the output of several orientation detectors are compared. Assuming that the same orientation detectors operate at high and low contrasts, the present results would argue in favor of the latter proposal. Detectors of broader bandwidth would result in coarser orientation discrimination at threshold[28] and the present results suggest that this is not the case. At suprathreshold levels, the outputs of many detectors can be compared, to produce optimal orientation discrimination.

A comparable phase discrimination task revealed an abnormality for discriminating local spatially band-pass stimuli that differed in phase (relative to the center of the Gaussian envelope) of 180°. More contrast was required by most of the amblyopes to discriminate these stimuli apart, compared with that needed to simply detect their presence. This deficit tended to increase with spatial frequency and ranged from approximately 6 dB in some subjects to 20 dB in others (CF, ST). Subsequent experiments revealed that although the deficit did not critically depend on the duration of stimulus presentation, for some amblyopes it depended on the absolute orientation and bar length of the stimulus. The polarity of stimuli with longer bars were harder (relative to their detection) to discriminate by the amblyopic visual system.

Several studies have shown that amblyopes exhibit anomalies for the encoding of the relative phase between different spatial components. Lawden et al.[18] showed this for a task in which observers had to discriminate between F and 3F sinusoids added in a peaks-add or peaks-subtract phase. Such a discrimination represents a 180° phase discrimination for the 3f component, and amblyopes required more contrast to do this task at high spatial frequencies relative to either the detection of the 3f component alone (termed simple detection threshold) or to the detection of the 3f component in the presence of the suprathreshold F component (termed compound detection). This suggests an anomaly of relative phase encoding for the 3f component beyond that of simple masking by the F component. Brettel et al.[20] demonstrated that amblyopes are insensitive to phase distortions in broadband images. Pass and Levi[19] showed that amblyopes need more contrast to discriminate the polarity of periodic ramp stimuli—a result that suggests that amblyopic eyes are deficient in detecting (i.e, in compound detection) and/or encoding the relative phase of the second harmonic component of these stimuli. In a subsequent study, Paul et al. showed that amblyopes have an anomaly in discriminating Cauchy functions of opposite polarity and that this anomaly increases with spatial frequency and could be as great as a sevenfold loss (Paul AD, et al. *IOVS* 1983;24:ARVO Abstract 24). This suggested an anomaly in the encoding of absolute phase. The present study using Gabors of

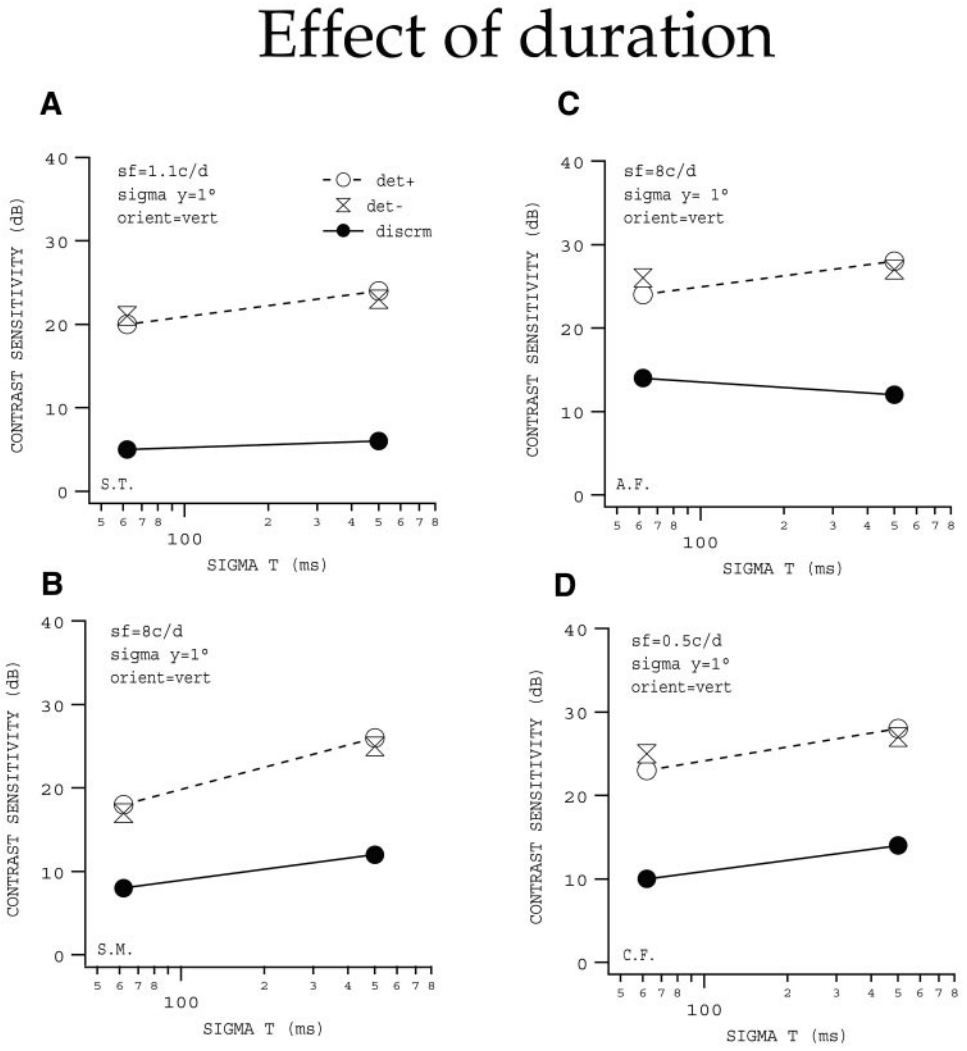

FIGURE 7. The effect of stimulus duration (sigma *t* of the Gaussian time envelope) on the phase discrimination anomaly for a selected high spatial frequency (relative to the amblyopic cutoff acuity) for four amblyopic eyes. Detection is compared with discrimination. Standard deviations are smaller than or equal to the symbol sizes.

different lengths, durations, and orientations confirms this earlier observation. We found comparable effects, ranging from a few decibels up to 20 dB (a factor of 10), in the contrast needed to detect the stimuli compared with that needed to identify their polarity.

Role of Eccentric Fixation

One possible confounding factor in this task is eccentric fixation. Most of our amblyopes exhibited eccentric fixation to some degree, and we wondered to what extent normal subjects could accomplish this type of discrimination using parafoveal vision. Figure 8 shows the effect of eccentric fixation for two normal observers on the simple detection and polarity discrimination of Gabor stimuli (1 and 5 cyc/deg). More contrast is needed by the parafoveal visual system to detect the stimulus—more for 5 cyc/deg compared with 1 cyc/deg. However the contrast polarity of the stimulus can be correctly discriminated close to detection threshold, and so this type of phase discrimination is not selectively impaired in the parafovea or midperiphery. Thus, it seems reasonable to conclude that the anomaly that we observed in our study of polarity discrimination in amblyopia is not a byproduct of the observer's eccentric fixation. This conclusion is further strengthened by the finding that two of our subjects (NN, SM) had central fixation and yet exhibited phase discrimination deficits. We conclude that there appears to be a separate phase discrimination deficit in amblyopia, one that is restricted to near threshold contrast and high spatial frequencies (i.e, relative to the cutoff of the amblyopic eye).

Relation to Suprathreshold Distortions

The finding that orientation discrimination at detection threshold is normal in amblyopia suggests that if the nonveridical perceptions in amblyopia are due to an anomaly in orientation processing,[12] then it is separate from the anomaly responsible for the elevated thresholds. However, it is hard to accept in the light of all the available evidence from positional tasks,[9,29–31] and now phase discrimination tasks[18–20,23,32] (Paul AD, et al. *IOVS* 1983;24:ARVO Abstract 24), that the nonveridical perceptions reported by some amblyopes do not have a spatial (i.e, phase or local positional) as well as an orientational component. Any model of the underlying disruption in amblyopia should consider not only orientational[12] but also positional disruptions to cortical processing in amblyopia. At present, it is unclear whether this phase anomaly is best explained at the level of individual cells or at the level of populations of cells. For example, on the one hand it may be due to a disruption in the geometrical properties of the excitatory and inhibitory inputs to individual, high spatial frequency, cortical cells driven by the amblyopic eye. On the other hand, it may be due to either a disruption in the topography of on- and off- cortical pathways or to the spatial accuracy with which this information can be accessed by later cortical areas.

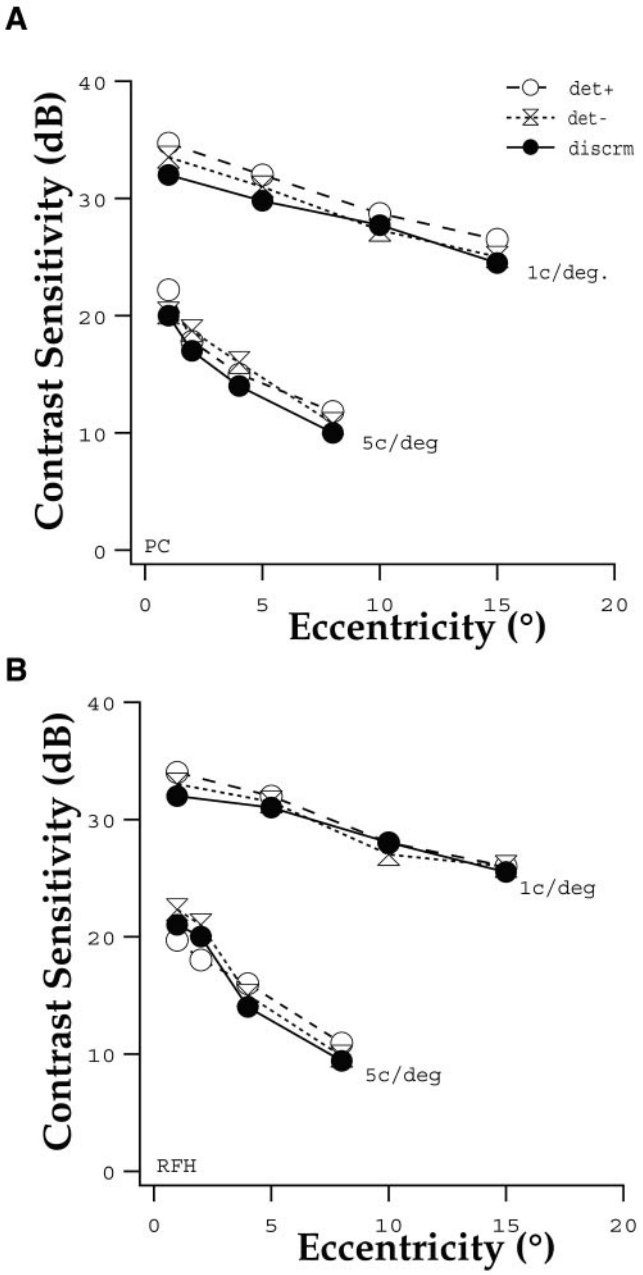

FIGURE 8. The effect of eccentric fixation on the contrast needed to detect and discriminate the local phase of a 1-cyc/deg and 5-cyc/deg Gabor stimulus for two normal observers. The standard deviations are smaller than or equal to the symbol sizes.

Threshold Versus Suprathreshold Deficit

Contrast thresholds can be elevated in amblyopic eyes without associated discrimination anomalies. This is clearly shown for orientation discrimination for all our amblyopic subjects. This suggests that the mechanisms responsible for raised thresholds are unlikely to be responsible also for the reported spatial distortions occurring with suprathreshold stimuli. Our current model for the threshold deficit in amblyopia is that cortical neurons in V1 that respond to high spatial frequencies have reduced contrast sensitivity[13] and spatial resolution.[15,16] Because no abnormality has been reported in other aspects of the amblyope's receptive field structure (i.e, orientation selectivity, phase selectivity) one is left to suppose that either a different population of cells are responsible for the suprathreshold distortions (e.g, those with higher thresholds) or it is how the outputs of these cells are analyzed that is responsible for the nonveridical spatial perceptions in amblyopia.

Acknowledgments

The authors thank the participants in the study for giving their time so generously, Pi-Chun Huang for help in the latter stages of the project, and David Ellemberg for comments on the manuscript.

References

1. Gstalder RJ, Green DG. Laser interferometric acuity in amblyopia. *J Pediatr Ophthalmol.* 1971;8:251–256.
2. Hess RF, Howell ER. The threshold contrast sensitivity function in strabismic amblyopia: Evidence for a two type classification. *Vision Res.* 1977;17:1049–1055.
3. Levi M, Harwerth RS. Spatio-temporal interactions in anisometropic and strabismic amblyopia. *Invest Ophthalmol Vis Sci.* 1977; 16:90–95.
4. Hess RF, Pointer JS. Differences in the neural basis if human amblyopias: the distribution of the anomaly across the visual field. *Vision Res.* 1985;25: 1577–1594.
5. Hess RF, Campbell FW, Zimmern R. Differences in the neural basis of human amblyopias: effect of mean luminance. *Vision Res.* 1980; 20:295–305.
6. Hess RF, Bradley A. Contrast coding in amblyopia is only minimally impaired above threshold. *Nature.* 1980;287:463–464.
7. Hess RF, Campbell FW, Greenhalgh T. On the nature of the neural abnormality in human amblyopia: neural aberrations and neural sensitivity loss. *Pflugers Arch Eur J Physiol.* 1978;377:201–207.
8. Bradley A, Freeman R. Is reduced vernier acuity in amblyopia due to position, contrast or fixation deficits? *Vision Res.* 1985;25:55–66.
9. Fronius M, Sireteanu R. Monocular geometry is selectively distorted in the central visual field of strabismic amblyopes. *Invest Ophthalmol Vis Sci.* 1989;30:2034–2044.
10. Lagreze WD, Sireteanu R. Two-dimensional spatial distortions in human strabismic amblyopia. *Vision Res.* 1991;31:1271–1288.
11. Sireteanu R, Lagreze WD, Constantinescu DH. Distortions in two-dimensional visual space perception in strabismic observers. *Vision Res.* 1993;33:677–690.
12. Barrett BT, Pacey IE, Bradley A, Thibos LN, Morrill P. Non-veridical visual perception in human amblyopia. *Invest Ophthalmol Vis Sci.* 2003;44:1555–1567.
13. Eggers HM, Blakemore C. Physiological basis of anisometropic amblyopia. *Science.* 1978;201:264–267.
14. Crewther DP, Crewther SG. Neural site of strabismic amblyopia in cats: spatial frequency deficit in primary cortical neurons. *Exp Brain Res.* 1990;79:615–622.
15. Kiorpes L, Kiper DC, O'Keefe LP, Cavanaugh JR, Movshon JA. Neuronal correlates of amblyopia in the visual cortex of macague monkeys with experimental strabismus and anisometropia. *J Neurosci.* 1998; 18; 6411–6424.
16. Kiorpes L, McKee SP. Neural mechanisms underlying amblyopia. *Curr Opin Neurobiol.* 1999;9:480–486.
17. Skottun BC, Bradley A, Freeman RD. Orientation discrimination in amblyopia. *Invest Ophthalmol Vis Sci.* 1986;30:532–537.
18. Lawden MC, Hess RF, Campbell FW. The discriminability of spatial phase relationships in amblyopia. *Vision Res.* 1982;22:1005–1016.
19. Pass AF, Levi DM. Spatial processing of complex stimuli in the amblyopic visual system. *Invest Ophthalmol Vis Sci.* 1982;23:780–786.
20. Brettel H, Caelli T, Hilz R, Rentschler I. Amblyopic processing of positional information. part III: sensitivity to phase distortion. *Exp Brain Res.* 1985;60:279–288.
21. Volkmann FC, Riggs LA, White KD, Moore RK. Contrast sensitivity during saccadic eye movements. *Vision Res.* 1978;18:1193–1199.
22. Robson JG, Graham N. Probability summation and regional variation in contrast sensitivity across the visual field. *Vision Res.* 1981;21:409–418.
23. Tolhurst DJ, Dealy RS. The detection and identification of lines and edges. *Vision Res.* 1975;15:1367–1372.

24. Demanins R, Wang Y.-Z, Hess RF. The neural deficit in strabismic amblyopia: sampling considerations. *Vision Res.* 1999;39:3575–3585.
25. Rentschler I, Hilz R. Abnormal orientation selectivity in both eyes of strabismic amblyopes. *Exp Brain Res.* 1979;37:187–191.
26. Vandenbussche E, Vogels R, Orban GA. Human orientation discrimination: changes with eccentricity in normal and amblyopic vision. *Invest Ophthalmol Vis Sci.* 1986;27:237–245.
27. Demanins R, Hess RF, Williams CA, Keeble DRT. The orientation discrimination deficit in strabismic amblyopia depends upon stimulus bandwidth. *Vision Res.* 1999;39:4018–4031.
28. Thomas JP, Gille J, Barker RA. Simultaneous visual detection and identification: theory and data. *J Opt Soc Am.* 1982;72:1642–1651.
29. Bedell HD, Flom MC. Monocular spatial distortion in strabismic amblyopia. *Invest Ophthalmol Vis Sci.* 1981;20:263–268.
30. Levi DM, Klein SA, Aitsebaomo AP. Vernier acuity, crowding and cortical magnification. *Vision Res.* 1985;25:963–977.
31. Hess RF, Holliday IE. The spatial localization deficit in amblyopia. *Vision Res.* 1992;32:1319–1339.
32. Kiper DC. Spatial phase discrimination in monkeys with experimental strabismus. *Vision Res.* 1994;34:437–447.